Bradydysrhythmias and Atrioventricular Conduction Blocks

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Bradydysrhythmias

Bradycardia is defined as a ventricular rate less than 60 beats per minute (bpm). Sinus bradycardia exists when a P wave precedes each QRS complex. This QRS complex is usually narrow (less than 0.120 seconds) because the impulse originates from a supraventricular focus (Fig. 1). On ECG, the P-P interval in sinus bradycardia closely matches the R-R interval, because the P wave is always preceding a QRS complex and the rate is regular. Each P wave within a given lead has the same morphology and axis, because the same atrial focus is generating the P wave.

There are specific incidences in which, despite the supraventricular focus, the QRS is widened (greater than 0.12 seconds). An example of this is a bundle branch block (right or left) in which the QRS complex is wide, but each QRS complex is still preceded by a P wave, and thus the underlying rhythm is still considered sinus bradycardia. Clues to differentiate this on ECG are that the PR interval usually remains constant and the QRS morphology is typical of a bundle branch block pattern.

Other ECG rhythms may seem like sinus bradycardia but in fact do not meet the definition as mentioned (see section on sinoatrial block).

Junctional rhythm is another example of a supraventricular rhythm in which the QRS complex morphology is usually narrow (less than 0.12 seconds) and regular. This is distinguished from sinus bradycardia on ECG because it is not associated with preceding P waves or any preceding atrial aberrant rhythms. On ECG, a junctional escape rate is usually 40–60 bpm, because the impulse is generated below the SA node, at the atrioventricular (AV) junction. A junctional rhythm with a rate slower than 40 bpm is termed

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doi:10.1016/j.emc.2005.08.006
emed.theclinics.com
junctional bradycardia, and a junctional rhythm with a rate faster than 60 bpm is termed an accelerated junctional rhythm or a junctional tachycardia; this reflects usurpation of pacemaker control from the sinus node (Fig. 2).

There are times when there are P waves evident on the ECG of patients who have a junctional rhythm, but unlike normal sinus rhythm or sinus bradycardia, these P waves are not conducted in an anterograde fashion. These are termed P’ waves and may appear before, during (in which case they are obscured), or after the QRS complex, depending on when the atrium is captured by the impulse emanating from the AV junction. Retrograde atrial capture is affected by the origin of the AV junctional impulse (physical location of the pacemaker, whether it is high, middle, or lower AV node) and the speed of conduction. As in sinus bradycardia, there are also times in which the QRS morphology is widened (greater than 0.12 seconds) because of a right or left bundle branch block.

Idioventricular rhythms are regular, but unlike sinus bradycardia or junctional rhythms, they are always characterized by a wide QRS complex (greater than 0.12 seconds), because their origin lies somewhere within the
ventricles (Fig. 3). On ECG, the rate is usually 20–40 bpm except for accelerated idioventricular rhythms (rate greater than 40 bpm).

Sinoatrial (SA) blocks result when there is an abnormality between the conduction of the impulse from the heart’s normal pacemaker (SA node) to the surrounding atrium. Because there is a wide range of severity of dysfunction, there are many ECG findings associated with SA blocks (also called SA exit blocks) (Fig. 4) [1]. As with AV block, SA block is characterized as first-, second-, and third-degree, with second-degree blocks subclassified as type I and type II.

First-degree SA block represents an increased time for the SA node’s impulse to reach and depolarize the rest of the atrium (ie, form a P wave). Because impulse origination from the SA node does not produce a deflection on the 12-lead ECG, there are no abnormalities seen on the 12-lead tracing with first-degree SA block.

Second-degree SA block is evident on the surface ECG. Second-degree SA block type I occurs when there is a progressively increasing interval for each SA nodal impulse to depolarize the atrial myocardium (ie, cause a P wave), which continues to lengthen until the SA node’s impulse does not depolarize the atrium at all. This is manifested by gradual shortening of the P-P interval with an eventual “dropped” P-QRS-T complex. It can be recognized by “grouped beatings” of the P-QRS-T complexes, or may manifest as irregular sinus rhythm (a sinus rhythm with pauses) on the ECG.

Second-degree SA block type II occurs when there is a consistent interval between the SA node impulse and the depolarization of the atrium with an occasional SA nodal impulse that is not conducted at all. On the ECG, there is a dropped P-QRS-T complex with a P-P interval surrounding the pause that is two to four times the length of the baseline P-P interval [2].

Second-degree SA block with 2:1 conduction is seen on ECG when every other impulse from the SA node causes atrial depolarization while the other is dropped. The ECG findings associated with this block are difficult. It is impossible to differentiate this from sinus bradycardia unless the beginning or termination of the SA block is caught on ECG. This manifests on ECG as a distinct halving (beginning) or doubling (termination) of the baseline rate.

Third-degree SA block occurs when none of the SA nodal impulses depolarize the atrium. This appears as a junctional rhythm with no P waves on the 12 lead tracing, because the focus now responsible for depolarization of the ventricles lies below the SA node. Sometimes there is a long pause on the ECG until a normal sinus rhythm is resumed. This pause is difficult
to distinguish from sinus pause or arrest. All pauses in SA blocks, however, should be a multiple (two to four times the length) of the P-P intervals on the ECG (see section on sinus pause/arrest for more details).

Sinus pause and sinus arrest are characterized by the failure of the SA node to form an impulse. Although sinus pause refers to a brief failure and a sinus arrest refers to a more prolonged failure of the SA node, there are no universally accepted definitions to differentiate the two. Because of this, they are often used interchangeably to describe the same cardiac event (Fig. 5) [3].

On ECG there is an absence of the P-QRS-T complex, resulting in a pause of undetermined length. Sinus pause may be preceded by any of these rhythms, the origin of which is in the atrium: sinus beats, ectopic atrial beats, and ectopic atrial tachycardia. Or it may appear on the ECG with
a junctional escape rhythm in which an AV nodal impulse has suppressed the sinus node [4]. After the sinus pause/arrest is seen on the ECG, the rhythm that follows also varies greatly. The sinus node most often resumes pacemaker activity and a normal sinus rhythm is seen. In cases in which it fails, however, the escape rhythm seen is usually from the AV node. If the AV node fails, the next pacemaker to take would result in an idioventricular rhythm. If all of these fail to generate an escape rhythm, the result is asystole.

The difficulty remains in distinguishing sinus pause/arrest from SA block. The biggest apparent difference between the two rhythms is the P-P interval. During sinus pause, the P-P interval is not a multiple of the baseline P-P interval. In SA block, however, the P-P interval should be a multiple of the baseline P-P interval.

Sinus arrhythmia is seen electrocardiographically as a gradual, cyclical variation in the P-P interval (Fig. 6). The longest P-P interval exceeds the shortest P-P interval by more than 0.16 seconds. Most commonly this occurs as a normal variation caused by respiratory variability; the sinus rate increases with inspiration and decreases during expiration [5]. In elderly individuals, it may be a manifestation of sick sinus syndrome.

Sick sinus syndrome is a collective term that includes a range of SA node dysfunction that manifests in various different ways on the ECG, including inappropriate sinus bradycardia, sinus arrhythmia, sinus pause/arrest, SA exit block, AV junctional (escape) rhythm (all discussed earlier), and the bradycardia-tachycardia syndrome. Bradycardia-tachycardia syndrome (or tachy-brady syndrome) is defined by bradycardic rhythms alternating with episodes of tachycardia. These tachycardic rhythms usually are supraventricular in origin but at times may be accelerated junctional or ventricular rhythms. A distinguishing finding of this syndrome on ECG, though difficult to capture, is the transition from the termination of the tachydysrhythmia
back to a sinus nodal rhythm. Often, severe sinus bradycardia, sinus pause/arrest, SA block, or junctional rhythm occur first until the sinus mechanism recovers (Fig. 7).

**Atrioventricular block**

Like SA block, AV block can be partial or complete and also is divided into first-, second-, and third-degree varieties. Second-degree, again similar to SA block, is divided into Mobitz type I (Wenckebach AV block) and Mobitz type II. A clue to differentiating between SA blocks and AV blocks is remembering where the conduction delay is occurring. In SA block, the dysfunction occurs between the SA node and the atrial myocardium; thus, there is a dropped P-QRS-T complex. In AV block, conduction is altered between the atrium and the ventricle, causing a prolonged PR interval and a dropped QRS-T complex (eventually a P wave occurs without a QRS-T behind it).

First-degree AV block is defined as a prolonged PR interval (greater than 0.20 seconds) that remains constant. The P wave and QRS complex have normal morphology, and a P wave precedes each QRS complex (Fig. 8). The lengthening of the PR interval results from a conduction delay from within the atrium, the AV node, or the His-Purkinje system. Most patients have a narrow QRS complex (less than 0.12 seconds), which indicates a block in the AV node, but occasionally there is a widened QRS complex associated with a delay in lower cardiac conduction. And as with SA blocks, patients may have a wide QRS complex caused by a coexisting bundle branch block.

Second-degree AV block, Mobitz type I is characterized by normal P wave and QRS complex morphology beginning with a PR interval that
lengthens progressively with each cycle until an impulse does not reach the ventricles and a QRS complex is dropped (Fig. 9). This block is usually at or above the AV node. On ECG, the PR interval lengthens as the R-R interval shortens. The R-R interval that contains the dropped beat is less than two of the shortest R-R intervals seen on the ECG. Also, on the ECG rhythm strip, a grouping of beats typically is seen, especially with tachycardia; this is referred to as “grouped beating of Wenckebach” [1,6]. All four of these ECG findings are typical of Mobitz type I block but unfortunately have been observed in less than 50% of all cases reported [1,7]. What has been reported are variations on all of the above, from PR intervals not lengthening progressively to conducting all atrial impulses to the ventricles [6,7]. These variations on second-degree Mobitz type I AV block seen on ECG do not change the clinical importance of this AV block [8].

Second-degree AV block, Mobitz type II is defined by constant PR intervals that may be normal or prolonged (>0.20 seconds). Unlike Mobitz type I second-degree AV block, however, Mobitz type II blocks do not demonstrate progressive lengthening of the PR interval on the ECG before a QRS complex is dropped. Also, unlike type I second-degree AV block, the QRS complex usually is widened, because the location of this block is often infranodal. The QRS complex may be narrow, however, indicating a more proximal location of block, usually in the AV node. The magnitude of the AV block can be expressed as a ratio of P waves to QRS complexes. For example, if there are four P waves to every three QRS complexes, it would be a 4:3 block (Fig. 10) [9].

Because Mobitz type II second-degree AV block does not have progressively lengthening PR intervals, differentiating type I from type II on ECG is simple, except in the case of 2:1 block. In second-degree AV block with 2:1
block, every other QRS-T is dropped (ie, two P waves for each QRS complex), so there is no opportunity to determine if the PR interval lengthens before the dropped QRS complex. If the ventricular beat is represented by a widened QRS complex, this suggests a more concerning Mobitz type II block, but ultimately it may be impossible to differentiate between the two. In that case, the physician should presume it is Mobitz type II, because it is more likely to progress to third-degree (complete) heart block.

High-grade or advanced AV block is a more clinically concerning variant of Mobitz type II block and is manifested by two or more P waves that are

Fig. 11. Third-degree AV block. Complete heart block is seen here with P waves (dots) that “march” through the QRS-T complexes; at times the P waves are obscured by these other waveforms. The atrial rate (approximately 90 bpm) is faster than the escape ventricular rate (approximately 60 bpm), which is driven by the junctional pacemaker; rephrased, the P-P interval is shorter than the R-R interval, as it should be in complete heart block. Note this patient is having an acute inferior myocardial infarction, with ST segment elevation (leads II, III, and aVF) and reciprocal ST segment depression (leads aVL and I). The right coronary artery is the culprit vessel.

Fig. 12. Complete heart block with periods of asystole. Note that several P waves occur at first without associated QRS complexes, before an idioventricular escape rhythm ensues. P waves are denoted by arrows. This patient survived and received an electronic pacemaker.
not conducted. This most often implies advanced conduction disease seen in anterior infarction and has high risk for progression to complete heart block [9]. On the ECG there are usually widened QRS complexes with ventricular rates between 20 and 40 bpm.

Third-degree AV block (complete heart block) occurs when no impulses from the atria reach the ventricles. The atria and ventricles thus are functioning independently (ie, there is AV dissociation), and the atrial rate is faster than the ventricular rate because the latter is an escape rhythm (Fig. 11). The escape rhythm controlling the ventricles is usually regular because of the increased autonomic control of the ventricle compared with the sinus node [10]. The atrial impulses (P waves) “march” out on the ECG, as do the ventricular depolarizations (QRS complexes), yet they are unrelated. The ventricular rate is usually 40–60 bpm with a narrow QRS complex when it is driven by a junctional pacemaker (within the AV node). If an infra-Hisian ventricular pacemaker takes over, the QRS complexes are wide and the rate is less than 40 bpm. Ventricular escape rhythms usually are associated with a poorer prognosis and are caused more commonly by acquired (non-congenital) conditions [6]. It is also possible that no escape rhythm is generated, resulting in asystolic arrest (Fig. 12).

References